INTERNAL INTESTINAL FISTULAE CAUSED BY AMOEBIASIS

A FIRST REPORT

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Though stricture of the colon has frequently been reported and is well known as a late complication of amoebiasis, there is no report in the literature of internal fistula caused by amoebiasis.

A recent experience at Baragwanath Hospital has prompted us to review the problem, and as a result we wish to present the following 4 cases of actual or impending amoebic internal fistula, in the form of a preliminary

report to further investigation which is proceeding.

CASE 1

S.Z., an adult African male, aged 49, was admitted to Baragwanath Hospital on 23 February 1957 with a history of several weeks of abdominal pain followed by severe diarrhoea and extreme weakness. He stated that he sustained an abdominal injury in December 1956, and dated the abdominal pain from that time.

Examination revealed a grossly dehydrated and emaciated patient with a generalized peritonitis. An X-ray of the abdomen showed gas under the diaphragm. The clinical diagnosis of amoebic perforation of the colon was not substantiated, in spite of the fact that 9 stool examinations were made during the next few days.

Meanwhile, on conservative therapy for generalized peritonitis, the patient made a slow recovery. A barium enema, performed after the acute episode had subsided, showed a colitis with enterocolic fistula (Fig. 1).

These findings in an African prompted a diagnosis of ulcerative colitis. Symptoms persisted with deterioration of the patient's condition, and a terminal ileostomy was carried out on 25 April as a desperate measure. The poor condition of the patient and the presence of a plastic peritonitis precluded an extensive laparotomy.

Postoperatively the patient's condition improved slightly, but after a further deterioration he died 10 days later. Autopsy showed multiple perforations of the colon with pericolic abscess formation, multiple adhesions, and enterocolic fistulae. On microscopic examination *Entamoeba histolytica* were found in sections taken from the colon.

Comment

This is a case of amoebic perforation of the colon with subsequent enterocolic fistula formation. The fact that amoebiasis was not considered as an aetiological factor in fistula formation led to misdiagnosis and the ultimate death of the patient.

It is of interest that, in a hospital where amoebiasis is fairly common, 9 separate stool examinations failed to reveal the pathogen.

Speculation about the part played by the abdominal injury in this rare complication of amoebiasis is of interest, but no conclusive deduction can be made.

CASE 2

J.K., an African male, aged 40, was admitted to Baragwanath Hospital on 20 March 1959 with a 10-week history of abdominal pain, continuous in nature and unrelated to food. There had been no vomiting or diarrhoea, but on occasion he had passed tarry stools.

At a small country hospital he was seen by a doctor who noted a mass in the left hypochondrium. A laparotomy was carried out and an inoperable tumour was diagnosed. No biopsy was performed and the abdomen was closed. He was then transferred to this hospital for further treatment.

Examination on admission revealed a wasted Bantu male with a mobile mass in the left hypochondrium, about 3 inches in diameter. Blood, stool and radiological investigations, including X-rays of chest and abdomen, pneumoperitoneum and intravenous pyelogram, proved unhelpful.

However, a barium enema showed an area of extreme narrowing of the left half of the transverse colon. The whole transverse colon was displaced downwards in a U-shaped curve (Fig. 2), and air-contrast studies showed a normal mucosal pattern in spite of the presence of an undilatable stricture.

A barium meal showed a normal gastric mucosal pattern, but along the greater curvature there was a long curved indentation suggestive of a mass distorting the stomach. Lateral and oblique films showed the stomach to be displaced anteriorly.

The conclusion drawn was that a tumour mass, either in the lesser sac or in the gastrocolic omentum, was present. The apparently normal colonic mucosal pattern associated with stricture of the colon remained a puzzling feature.

Following intestinal sterilization, operation was undertaken on 27 May, and a mass involving the greater curvature of the stomach, the greater omentum and the transverse colon was found. A resection, including both greater curvature and

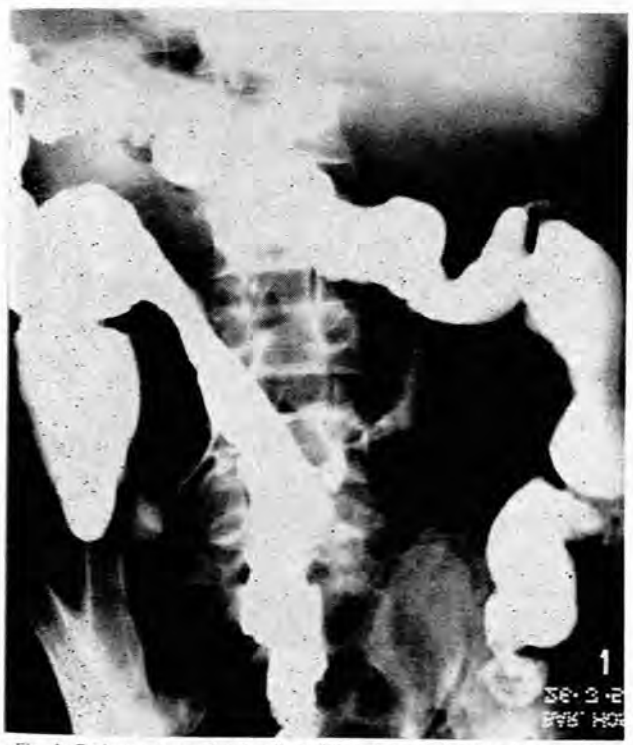

Fig. 1. Barium enema showing ileal filling through the ileo-caecal valve, an entero-colic fistula to the left of the ileum, and a blind fistula to the right of the ileum (case 1).

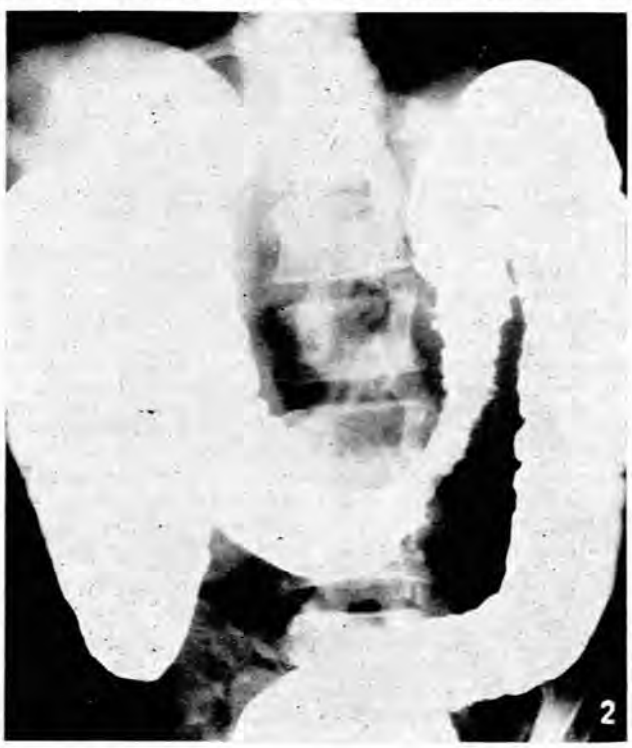

Fig. 2. Barium enema showing narrowing and indentation of the transverse colon by the amoeboma (case 2).

transverse colon, was carried out and the patient made an uneventful recovery.

Histology of the operation specimen revealed the typical features of an amoeboma, in which *Entamoeba histolytica* was found. There was fairly advanced necrosis of the central portion of the amoeboma and the parts adjacent to the stomach and colon, and subsequent fistula at this site seemed inevitable.

Comment

This is a case in which an amoeboma of the transverse colon spread to the stomach *via* the gastrocolic omentum. Central necrosis of the amoeboma seemed to herald fistula at this site.

The recovery of this patient was entirely uneventful, in spite of the fact that anti-amoebic therapy was started only 10 days after the operation, subsequent to the histological report.

CASE 3

A.K., an African male, aged 32, was admitted on 19 July 1959 with a 5-week history of severe pain in the left hypochondrium associated with a bloody diarrhoea. The pain was occasionally relieved by defaecation, but only temporarily. His appetite was poor and there had been a weight-loss of 14 lb. Examination revealed a somewhat ill-looking male subject whose mucosae were pale.

Further positive findings were a 2-finger non-tender enlargement of the liver, and an irregular longitudinal mass extending from the left hypochondrium down into the pelvis. This was moderately tender and relatively mobile in the transverse direction only.

An amoeboma of the descending colon was diagnosed, and this was confirmed when both cystic and vegetative forms of *Entamoeba histolytica* were found on stool examination.

Anti-amoebic therapy with emetine by injection, chloroquine, and 'aureomycin' was started forthwith. Within 24 hours of the beginning of therapy, signs of a generalized peritonitis became established and a perforation of the amoeboma was diagnosed. Conservative therapy with nasogastric suction and intravenous fluids was then instituted. The emetine by injection was continued, and the aureomycin was given in the drip. By the tenth day the peritonitis had resolved and the mass was no longer palpable, but there was some residual distension of the abdomen. Further progress was complicated by a deep-calf-vein thrombosis which was treated with anticoagulants.

In spite of a complete course of anti-amoebic therapy, including chloroquine, the patient continued having diarrhoea and failed to gain weight in the face of vigorous supportive measures. Further stool examinations, sigmoidoscopy and biopsy were negative for amoebae. Nevertheless, a further full course of anti-amoebic therapy was instituted 1 month after the first one, without any effect.

While on this régime a barium enema was performed on 6 October. An enterocolic fistula was demonstrated between the splenic flexure and the upper jejunum, the barium flowing rapidly into the duodenum, stomach and oesophagus (Fig. 3).

The patient's condition then deteriorated rapidly, and because of marked mental disturbance, treatment became difficult to carry out. He would not tolerate the intravenous infusion, and an emergency transverse colostomy was carried out under local anaesthesia in an attempt to alleviate the diarrhoea. However, he died the day after operation.

Autopsy revealed multiple pulmonary emboli and a jejunocolic fistula, about 8 inches distal to the duodenojejunal flexure, communicating with the splenic flexure.

Histological examination of tissue taken at this and other sites in the colon showed no evidence of amoebiasis, demonstrating the adequacy of the anti-amoebic therapy.

Comment

This was a case of amoebic enterocolic fistula following perforation of an amoeboma. The clinical course of this patient's illness reflects a possible sequence of events in fistula formation from amoebiasis.

Since the site of the fistula was in the region of the amoeboma, it is suggested that perforation of the amoeboma into small bowel led to fistula formation.

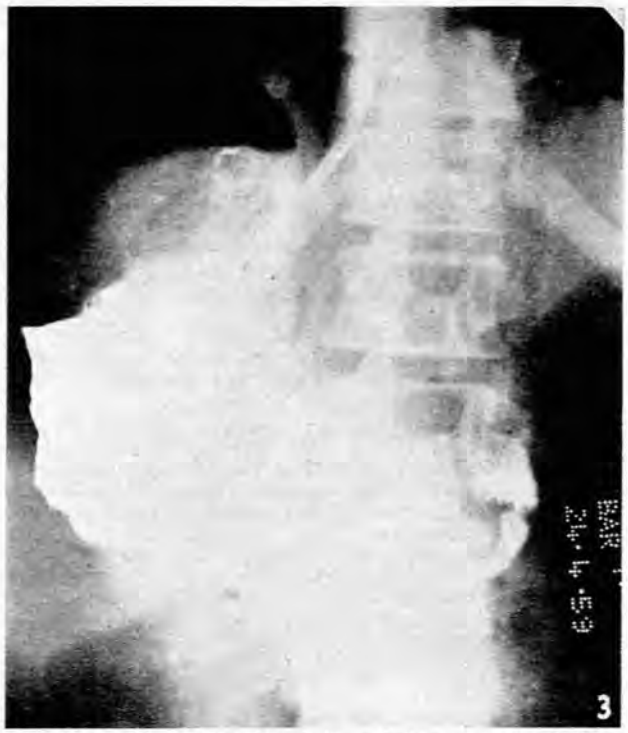

Fig. 3. An oblique view taken during a barium-enema examination, showing gastric and oesophageal filling (case 3).

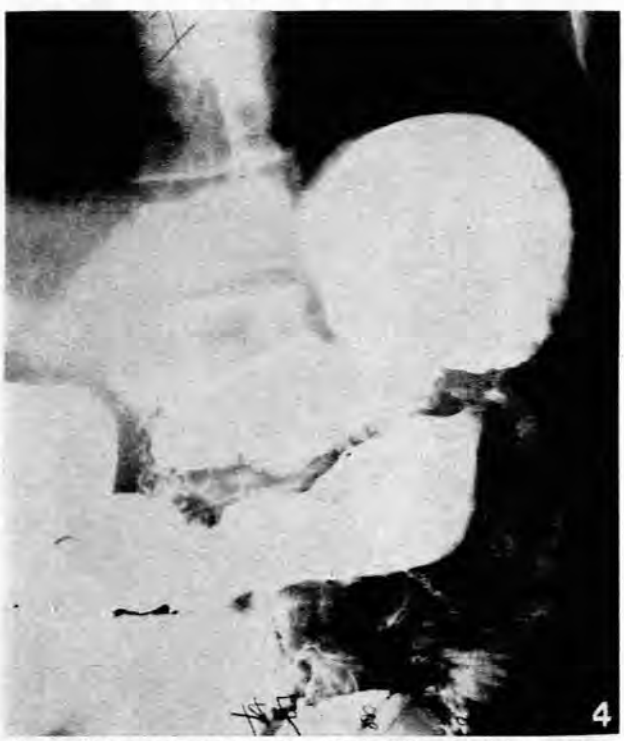

Fig. 4. Barium meal demonstrating colonic filling caused by a gastrocolic fistula (case 4).

CASE 4

J.M., a 44-year-old African male, underwent a full course of anti-amoebic therapy in hospital in July 1959 for proved amoebic dysentery. Five days after discharge he complained

of abdominal pain with severe bloody diarrhoea. His appetite remained good, but as soon as he ate he experienced pain and went to stool. He was re-admitted to Baragwanath Hospital on 30 August, suffering from the above symptoms and extreme weakness.

Examination revealed a cachectic, severely dehydrated patient with a generalized peritonitis. On rectal examination there was considerable spasm of the anal sphincter, and on withdrawing the finger the glove was found to be soiled with anchovy-like material.

He improved slightly on conservative therapy without anti-amoebic drugs, but the symptoms persisted much as before. A diagnosis of gastrocolic fistula was made and this was confirmed on barium-meal examination (Fig. 4).

Following preparation with blood and plasma, a proximal-loop colostomy was carried out with immediate improvement. While preparing the patient for further surgery a recrudescence of diarrhoea, with entamoebae in the stools, occurred. In spite of vigorous anti-amoebic therapy, the infection was not controlled and the patient died within 3 weeks. Permission for a postmortem examination was not obtained.

Comment

This was a patient with fulminating amoebic dysentery, who developed peritonitis in the course of his illness, probably following perforation of the colon. We feel that a pericolic abscess developed as a result of the peritonitis, and that, as in case 3 where an amoeboma was responsible for an entero-colic fistula, rupture of this abscess into the stomach resulted in a gastrocolic fistula.

DISCUSSION

Many reports on the sequelae of amoebiasis indicate that a small proportion of patients are left with chronic colitis and recurrent diarrhoea.

It is our contention, however, that some of these patients with residual diarrhoea have internal fistulae, with the production of blind-loop syndromes. Schlesinger[1] and Cameron *et al.*[2] have also shown that any lesion, such as a stricture, which produced stagnation and bacterial proliferation in some part of the gastro-intestinal tract, also produced a syndrome with the same features as the blind-loop syndrome. The common features in both these types of case is bacterial proliferation, and the frequent presence of bacteria in the normally sterile upper small bowel.

We suggest that, in the chronic diarrhoea persisting after adequately treated amoebiasis, investigation of the gastro-intestinal tract, to eliminate either stricture or fistula as a cause, should be carried out.

Where the blind-loop syndrome is operative, excellent symptomatic improvement is obtained by intestinal sterilization, and this should be used as an adjunct to surgery.

SUMMARY

1. A first report of amoebiasis causing internal intestinal fistulae is presented.

2. Four cases are described, each with interesting features giving rise to speculation on the possible pathogenesis of internal fistulae, viz.:

(*a*) Central necrosis of an amoeboma adherent to 2 hollow viscera, and breakdown of the abscess.

(*b*) Perforation of an amoeboma with subsequent adhesion of small bowel to the colon.

(*c*) Perforation followed by a generalized peritonitis may result in a residual abscess lying between hollow viscera, and breakdown of the abscess subsequently may cause fistula formation.

3. It is suggested that the persisting diarrhoea which complicates the recovery of adequately treated amoebiasis may arise because of the occurrence of stricture or a blind-loop syndrome.

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